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WHAT IS CLAIMED IS:

1. A method of treating pathologic cardiac hypertrophy and heart failure comprising:

   (a) identifying a patient having pathologic cardiac hypertrophy; and

   (b) administering to said patient a histone deacetylase inhibitor.

2. The method of claim 1, wherein said histone deacetylase inhibitor is selected from the group consisting of trichostatin A, trapoxin B, MS 275-27, m-carboxycinnamic acid bis-hydroxamide, depudecin, oxamflatin, apicidin, suberoylanilide hydroxamic acid, Scriptaid, pyroxamide, 2-amino-8-oxo-9,10-epoxy-decanoyl, 3-(4-aroyl-1 *H*-pyrrol-2-yl)-*N*-hydroxy-2-propenamide and FR901228.

3. The method of claim 1, wherein administering comprises intravenous administration of said histone deacetylase inhibitor.

4. The method of claim 1, wherein administering comprises oral, transdermal, sustained release, suppository, or sublingual administration.

5. The method of claim 1, further comprising administering to said patient a second therapeutic regimen.

6. The method of claim 5 wherein said second therapeutic regimen is selected from the group consisting of a beta blocker, an iontrope, diuretic, ACE-I, AII antagonist, and Ca^{++}-blocker.

7. The method of claim 5, wherein said second therapeutic regimen is administered at the same time as said histone deacetylase inhibitor.

8. The method of claim 5, wherein said second therapeutic regimen is administered either before or after said histone deacetylase inhibitor.

9. The method of claim 1, wherein treating comprises improving one or more symptoms of cardiac hypertrophy.

10. The method of claim 9, wherein said one or more symptoms comprises increased exercise capacity, increased blood ejection volume, left ventricular end diastolic pressure, pulmonary capillary wedge pressure, cardiac output, cardiac index, pulmonary artery pressures, left ventricular end systolic and diastolic dimensions, left and right ventricular wall stress, or wall tension, quality of life, disease-related morbidity and mortality.

11. A method of preventing pathologic cardiac hypertrophy and heart failure comprising:

(a) identifying a patient at risk of developing pathologic cardiac hypertrophy; and
(b) administering to said patient a histone deacetylase inhibitor.

12. The method of claim 11, wherein said histone deacetylase inhibitor is selected from the group consisting of trichostatin A, trapoxin B, MS 275-27, m-carboxycinnamic acid bis-hydroxamide, depudecin, oxamflatin, apicidin, suberoylanilide hydroxamic acid, Scriptaid, pyroxamide, 2-amino-8-oxo-9,10-epoxy-decanoyl, 3-(4-aroyl-1 *H*-pyrrol-2-yl)-*N*-hydroxy-2-propenamide and FR901228.

13. The method of claim 11, wherein administering comprises intravenous administration of said histone deacetylase inhibitor.

14. The method of claim 11, wherein administering comprises oral, transdermal, sustained release, suppository, or sublingual administration.

15. The method of claim 11, wherein the patient at risk may exhibit one or more of comprises long standing uncontrolled hypertension, uncorrected valvular disease, chronic angina and/or recent myocardial infarction.

16. A method of identifying inhibitors of cardiac hypertrophy comprising:

(a) providing a histone deacetylase inhibitor;
(b) treating a myocyte with said histone deacetylase inhibitor; and
(c) measuring the expression of one or more cardiac hypertrophy parameters,

wherein a change in said one or more cardiac hypertrophy parameters, as compared to one or more cardiac hypertrophy parameters in a myocyte not treated with said histone deacetylase inhibitor, identifies said histone deacetylase inhibitor as an inhibitor of cardiac hypertrophy.

17. The method of claim 16, wherein said myocyte is subjected to a stimulus that triggers a hypertrophic response in said one or more cardiac hypertrophy parameters.

18. The method of claim 17, wherein said stimulus is expression of a transgene.

19. The method of claim 17, wherein said stimulus is treatment with a drug.

20. The method of claim 16, wherein said one more cardiac hypertrophy parameters comprises the expression level of one or more target genes in said myocyte, wherein expression level of said one or more target genes is indicative of cardiac hypertrophy.

21. The method of claim 20, wherein said one or more target genes is selected from the group consisting of ANF, α-MyHC, β-MyHC, α-skeletal actin, SERCA, cytochrome oxidase subunit VIII, mouse T-complex protein, insulin growth factor binding protein, Tau-microtubule-associated protein, ubiquitin carboxyl-terminal hydrolase, Thy-1 cell-surface glycoprotein, or MyHC class I antigen.

22. The method of claim 20, wherein the expression level is measured using a reporter protein coding region operably linked to a target gene promoter.

23. The method of claim 22, wherein said reporter protein is luciferase, β-gal, or green fluorescent protein.

24. The method of claim 20, wherein the expression level is measured using hybridization of a nucleic acid probe to a target mRNA or amplified nucleic acid product.

25. The method of claim 16, wherein said one or more cardiac hypertrophy parameters comprises one or more aspects of cellular morphology.

26. The method of claim 25, wherein said one or more aspects of cellular morphology comprises sarcomere assembly, cell size, or cell contractility.

27. The method of claim 16, wherein said myocyte is an isolated myocyte.

28. The method of claim 16, wherein said myocyte is comprised in isolated intact tissue.

29. The method of claim 16, wherein said myocyte is a cardiomyocyte.

30. The method of claim 29, wherein said cardiomyocyte is located *in vivo* in a functioning intact heart muscle.

31. The method of claim 30, wherein said functioning intact heart muscle is subjected to a stimulus that triggers a hypertrophic response in one or more cardiac hypertrophy parameters.

32. The method of claim 31, wherein said stimulus is aortic banding, rapid cardiac pacing, induced myocardial infarction, or transgene expression.

33. The method of claim 31, wherein said one or more cardiac hypertrophy parameters comprises right ventricle ejection fraction, left ventricle ejection fraction, ventricular wall thickness, heart weight/body weight ratio, or cardiac weight normalization measurement.

34. The method of claim 16, wherein said one or more cardiac hypertrophy parameters comprises total protein synthesis.

35. A method of identifying inhibitors of cardiac hypertrophy comprising:

(a) providing at least one class I and one class II histone deacetylase;

(b) contacting said histone deacetylases with a candidate inhibitor substance; and

(c) measuring the activity of said histone deacetylases,

wherein a greater decrease in class I histone deacetylase activity than class II histone deacetylase activity identifies said candidate inhibitor substance as an inhibitor of cardiac hypertrophy.

36. The method of claim 35, wherein said histone deacetylases are purified away from whole cells.

37. The method of claim 35, wherein said histone deacetylases are located in an intact cell.

38. The method of claim 35, wherein said cell is a myocyte.

39. The method of claim 38, wherein said myocyte is a cardiomyocyte.

40. The method of claim 35, wherein said class I histone deacetylase is selected from the group consisting of HDAC1, HDAC2, HDAC3, and HDAC 8.

41. The method of claim 35, wherein said class II histone deacetylase is selected from the group consisting of HDAC4, HDAC5, HDAC6, HDAC7, HDAC 9, and HDAC 10.

42. The method of claim 35, wherein the activity of more than one class I histone deacetylase is measured.

43. The method of claim 35, wherein the activity of more than one class II histone deacetylase is measured.

44. The method of claim 35, wherein the activity of more than one class I histone deacetylase and more than one class II histone deacetylase is measured.

45. The method of claim 35, wherein said candidate inhibitor substance has inhibitory activity against at least one class I histone deacetylase and has no activity against at least one class II histone deacetylase.

46. The method of claim 45, wherein said candidate inhibitor substance has inhibitory activity against multiple class I histone deacetylases and has no activity against multiple class II histone deacetylases.

47. The method of claim 35, wherein said candidate inhibitor substance has inhibitory activity against at least one class I histone deacetylase that is at least two-fold greater than its inhibitory activity against at least one class II histone deacetylase.

48. The method of claim 47, wherein said candidate inhibitor substance has inhibitory activity against at least one class I histone deacetylase that is at least five-fold greater than its inhibitory activity against at least one class II histone deacetylase.

49. The method of claim 35, wherein measuring HDAC activity comprises measuring release of a labeled acetyl group from a histone.

50. The method of claim 49, wherein said acetyl group is labeled with a radiolabel, a fluorescent label or a chromphore.